Disorders of Sodium

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KEYWORDS

• Hypernatremia • Hyponatremia • Sodium • Hypertonic saline
• Osmotic demyelination syndrome • SIADH

KEY POINTS

- Evaluating the patient's volume status is critical to helping ascertain the possible cause for the sodium imbalance.
- Rapidity of the development of the patient's sodium imbalance and if the patient has symptoms will often drive the management of the patient.
- Patients with severe symptoms require rapid treatment to prevent permanent neurologic sequelae.
- If hypertonic saline therapy is not readily available, you can use 8.4% sodium bicarbonate as an acceptable alternative.
- Preventing overcorrection should be foremost on the emergency clinician/s mind when managing sodium imbalance. Have a low threshold to check sodium levels more frequently while correcting.

INTRODUCTION

Dysnatremias are one of the most common electrolyte disturbances that present to the emergency department, with hyponatremia being the most common electrolyte abnormality in the United States. Between 3 and 6 million patients per year present to a clinical setting with hyponatremia and somewhere between 12% and 20% of patients admitted from the emergency department have a sodium imbalance.[1,2] One study found that hypernatremia was found in 2% of patients and hyponatremia was found in 10% of patients presenting to the emergency department.[3] Severe hypernatremia and hyponatremia are associated with considerable morbidity and mortality, and even mild hyponatremia is associated with worse outcomes when it complicates other conditions such as heart failure.[4]

Hyponatremia is defined as a serum sodium level of less than 135 mmol/L, and hypernatremia is defined as a serum sodium level of greater than 145 mmol/L. Because sodium is the most abundant cation in extracellular fluid, it is the primary

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Emerg Med Clin N Am 41 (2023) 697–709
https://doi.org/10.1016/j.emc.2023.06.003
0733-8627/23/Published by Elsevier Inc.
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determinant of plasma osmolality. As a result, any derangements in sodium can affect the balance of cations, anions, and overall plasma osmolality resulting in potential ramifications clinically, specifically the central nervous system (CNS).

The difference between a minor symptom and a life-threatening condition caused by an imbalance of sodium is often a consequence of the rapidity of change in sodium concentration, not necessarily the overall deficit. Severity of sodium imbalance depends on the patient's symptoms because the serum level does not always correlate to the patient's symptoms. Therefore, it is important to understand that, although total-body salt content may be abnormal, the vast majority of dysnatremias originate from a primary imbalance in electrolyte-free water intake and loss. The perturbation in water balance rather than the salt content is commonly the problem.

Physiology

The body maintains a delicate balance of water for its physiologic processes. Ultimately, the body's water is governed by solute concentrations or tonicity, particularly sodium, which serves as a marker for plasma tonicity. When this balance sways and sodium concentration changes, the CNS is often affected first and the most significantly. Moreover, depending on the rate of change, symptoms can range anywhere from asymptomatic to catastrophic and life threatening.

Plasma osmolality is a ratio of plasma solutes and plasma water. Simply, it can be estimated as follows:

$$Posm = 2[Na] + \frac{[Glucose]}{18} + \frac{[BUN]}{2.8}$$

Plasma tonicity factors only those solutes that do not easily traverse across cell membranes (ie, sodium salts); therefore, this can be thought of as the effective plasma osmolality. Even more simply, it is the same equation without factoring in urea, as urea also equilibrates freely across cell membranes.

$$\text{Plasma } tonicity = 2[Na] + \frac{[Glucose]}{18}$$

Plasma tonicity, or the effective plasma osmolality, is largely regulated by the renal system, antidiuretic hormone (ADH), and the thirst response to regulate relative free water intake. The regulation of salt and water can be accomplished independently as one can consume foods with increased or decreased salt and/or drink water. In order to maintain the physiologically normal range of 275 to 295 mOsm/L of water, the posterior pituitary gland is highly sensitive to osmolality fluctuations and may secrete ADH.[5] More specifically, the hypothalamic nuclei are sensitive to changes in serum osmolality within 1% to 2%.[5] Because serum osmolality decreases (this is most often in cases of polydipsia), the posterior pituitary will not secrete ADH, the nephrons will not resorb free water, and the urine will become more diluted (ie, increased urine osmolality).[5,6] Conversely, because serum osmolality increases (ie, in cases of dehydration), the posterior pituitary will secrete ADH, which causes a multitude of metabolic effects in order to increase water reabsorption and decrease diuresis, thus concentrating the urine. ADH affects increased expression of aquaporin-2 receptors to capture water in the luminal collecting ducts of the nephrons. Furthermore, through vasoconstriction, ADH can decrease blood flow in the renal medulla. This allows for an increased hypertonicity surrounding the loop of Henle, increasing water reabsorption.[7] Other factors may also stimulate ADH release. These include hypovolemia, drugs, pain, emotional stress, nausea, pregnancy, menstruation, hypoglycemia, severe

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hypoxemia and hypercapnia, and distribution into the body's "third space" (ie, sepsis, severe burns, trauma).[6,8–10]

Finally, although ADH regulates water retention, the body's thirst mechanism can control how much free water is allowed into system. Separate from diuresis, whether through ventilation, evaporation of sweat for bodily cooling, or water lost in stool, the body will also lose free water through insensible losses. This is estimated to be 40 to 800 mL per day.[11] Thirst prevents against dehydration more so than ADH.[6] Its threshold is a slightly higher threshold of plasma osmolality than ADH.[12] In this way, one can view thirst as the marker for the upper limit of osmolality and ADH release the lower limit. Proper functioning of these mechanisms optimizes the body's fluid balance and maintains water homeostasis. Therefore, anything that interferes with either function disrupts the delicate fluid balance and leaves the patient to risks of developing hyponatremia and hypernatremia and the subsequent symptoms associated with them.

HYPONATREMIA

Defined as a serum sodium level less than 135 mmol/L, hyponatremia is the most common electrolyte disorder found in any medical setting.[13] In cases of hyponatremia, the most common cause is hemodilution rather than loss of total sodium. Specifically, it is most commonly due to a failure to secrete free water. The emergency clinician ought to recognize and respect this pathologic condition because even the mild cases of hyponatremia can increase mortality in patients both inpatient and outpatient and increase morbidity in hospitalized patients.[14–17]

Presentation

Patients who present with hyponatremia have variable clinical presentations. Although hyponatremia often presents asymptomatically, hyponatremia with sodium less than 125 mmol/L often present with signs and symptoms that reflect neurologic dysfunction usually due to cerebral edema.[18,19] The severity of symptoms typically depends on the rate of reduction rather than the serum sodium level. Common complaints are usually broken into 2 categories: mild and severe. Mildly symptomatic patients commonly complain of headache, nausea, vomiting, lethargy, and confusion. Untreated, it may progress severe symptoms that include severe confusion, seizure, coma, and death.

In cases of chronic hyponatremia, the brain has 2 steps in protecting itself, thus decreasing the severity of symptoms. First, cerebral edema causes an increased interstitial hydrostatic pressure, which yields a net flow of fluid from the brain tissue and into the cerebrospinal fluid.[20] Second, in its attempt to prevent cerebral edema, the brain will draw out other solutes in an effort to neutralize the osmotic gradient and decrease swelling.[4] Patients with sodium concentrations less than 120 mmol/L most commonly present with nausea and vomiting, which is not usually associated with severe adverse outcomes.[21] However, when presenting with acute hyponatremia or acute on chronic cases, nausea, and vomiting can portend life-threatening cerebral edema.

Evaluation and Diagnosis

Making the diagnosis comes from evaluating the patient's serum sodium level from bloodwork, usually a basic metabolic panel or comprehensive metabolic panel. Once the emergency clinician recognizes a patient is hyponatremic, the next step is to determine the possible cause for hyponatremia. Common causes of hyponatremia

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are found in **Table 1**. Additionally, it is important to ascertain the patient's volume status and plasma osmolality because true hyponatremia is due to a hypoosmolar state. Cases of hypertonic hyponatremia occur when serum osmolality exceeds 290 mOsm/kg and isotonic hyponatremia between 275 and 290 mOsm/kg. Hypertonic hyponatremia is most often due to another solute increasing, contributing to the calculated number, such as cases of severe hyperglycemia; these solutes too act as osmotic forces causing fluid shifts. In cases such as diabetic ketoacidosis or hyperosmolar hyperglycemia, the abundance of serum glucose acts as an osmotic force, drawing free water out of the cell and into the extracellular space, and thus decreasing the relative concentration of serum sodium while increasing serum tonicity. Other common causes include administration of hypertonic agents, such as mannitol or hypertonic saline (HTS), which are critical in the acute management of patients with sudden increases of intracranial pressure, that is, traumatic intracranial hemorrhage and hemorrhagic strokes.[22] Given the patient's plasma osmolality and calculated osmolality, the clinician can then determine if there is a concurrent osmolar gap at play. The presence of a wide osmolar gap will lead to osmotic diuresis in the patient and further decreased plasma sodium concentrations.

In normal patients, the plasma water consists of approximately 93% of the volume, with the remaining 7% being attributed to fats and proteins.[23] Isotonic hyponatremia can be seen in pseudohyponatremia, which is a laboratory artifact in processing the blood sample for its sodium concentration. Most commonly, the cause is due to severely elevated cholesterol.[24] Other causes of pseudohyponatremia include hypertriglyceridemia, obstructive jaundice, which can cause extreme levels of cholesterol and lipoprotein-X, and myeloma and subsequent hyperproteinemia.[25,26] These patients will have unchanged concentrations of plasma water sodium and osmolality. They will largely be asymptomatic with respect to their hyponatremia and require no acute or emergent interventions.

Most commonly, hyponatremia is seen in hypotonic states. Hypotonic hyponatremia will have serum osmolality less than 275 mOsm/kg. There are several types of hypotonic hyponatremia: hypervolemic, euvolemic, and hypovolemic hyponatremia. It is important to note that other classification systems also exist, such as classifications according to the body's ability to excrete dilute urine. Typically, measurement of urine sodium can elucidate the hypotonic hyponatremia variant, that is, high urine sodium generally points to hemodilution as the culprit.[8] Therefore, the variant is likely hypovolemic hyponatremia. However, given the setting of the emergency department, volume status can be assessed quickly to provide further medical decision-making. For example, a dehydrated or hypovolemic patient's kidneys will respond by retaining

Table 1
Common causes of hyponatremia

Hypervolemic hyponatremia	Euvolemic hyponatremia SIADH	Hypovolemic hyponatremia
Heart failure	Drugs	Vomiting
Hepatic failure	Primary polydipsia	Diarrhea
Renal failure	Beer potomania	Exercise induced
Nephrotic syndrome	Hyperglycemia	Diuretics (thiazides most commonly)
	Hypothyroidism	Cerebral salt wasting syndrome
	Adrenal insufficiency	
	Hypokalemia	
	Diet	

Abbreviation: SIADH, syndrome of inappropriate antidiuretic hormone excretion.

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sodium and the water that follows. Consequently, urine sodium should be lower. Therefore, in states of hypotonic hyponatremia, if the urine sodium is greater than 20 mEq/L, the likely culprit is nephrogenic in etiology. Initial workup should also include the comprehensive metabolic panel, thyroid stimulating hormone (TSH), cortisol, and urine sodium. As discussed above, when the body has a low effective circulating volume, that is, a state of intravascular depletion, it will retain water thus concentrating the urine. When there is impairment of water excretion, urine osmolality will be elevated.[23] Except in cases of polydipsia and the "tea and toast" diet or beer potomania, wherein the urine osmolality will be less than 100 mOsm/kg, hyponatremic states will typically be greater than 200 mOsm/kg. With this methodology, one can elucidate or at least narrow down the differential diagnosis.

In states such as congestive heart failure, liver failure and subsequent cirrhosis, and nephrotic syndrome, the effective arterial blood volume is decreased due to decreased cardiac output, congestion with subsequent vasodilation, or protein loss.[27] ADH is secreted in response to decreased osmolality, causing sodium and water reabsorption while decreasing free water secretion. These patients will "third space" the fluids, mainly redistributing the fluid into the interstitial space and cause hypervolemic hyponatremia. They will present with edema and seem "volume up."

In hypovolemic hyponatremia, patient will seem dry or "volume down" on examination. They will often lack edema on examination and skin turgor may be poor. If the urine sodium is elevated, this may be due to renal losses in cases of diuretic use, salt wasting, or adrenal insufficiency. If it is decreased, one should suspect extrarenal losses, as in cases of gastroenteritis.

In euvolemic hyponatremia, which is the most common variant of true hyponatremia, evaluation of volume status can be unclear. Most of these cases are due to syndrome of inappropriate antidiuretic hormone excretion (SIADH). Other causes include psychogenic polydipsia and drug-induced hyponatremia. In some disease states, ADH may be secreted despite normal or increased plasma volume. Common conditions manifesting as SIADH include malignancy, central nervous system disorders, drugs, HIV, tuberculosis, and pneumonia.[27] However, SIADH is a diagnosis of exclusion. Since Bartter and Schwartz first described the syndrome, the diagnostic criteria have not changed: hypoosmolality (<275 mosm/kg); hyperosmolar urine (>100 mosm/kg); euvolemia; increased urine sodium (>20 mEq/L); and no diuretic use or evidence of thyroid or adrenal disease.[28]

Management

Management depends on the patient's symptoms and rapidity of development of the hyponatremia, if able to be elicited from the patient's history. Acute hyponatremia is defined as a sodium level of less than 135 mmol/L that develops during a period of 48 hours. The short course is not enough time for cells to adapt to the new extracellular tonicity, which may result in cerebral edema. If it is known that the hyponatremia has been present longer than 48 hours, then it is considered chronic.[13] Chronic hyponatremia, however, is oftentimes asymptomatic because the cells have had time to adapt to the extracellular environment. Intervening may cause the dreaded complication of osmotic demyelination syndrome (ODS).[13]

The management of hyponatremia should be individualized based on the patient's specific clinical condition, underlying causes, and comorbidities. The approach to treatment of hyponatremia depends on multiple factors, which includes duration, severity (degree), and severity of symptoms. Concentrations between 130 and 134 mEq/L and between 120 and 129 mEq/L are defined as mild and moderate

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hyponatremia, respectively. Once concentrations decline less than 120 mEq/L, it is considered severe hyponatremia.[13]

Especially in cases of chronic hyponatremia, some patients may present asymptomatically or with vague symptoms despite having moderate-to-severe hyponatremia. Although the acuity and the degree of sodium derangement are both important considerations for the clinician, guidelines now support that emergent intervention is more governed by the severity of the patient's symptoms. Mild-to-moderate hyponatremia may have relatively nonspecific symptoms to include headache, fatigue, nausea, vomiting, dizziness, gait disturbances, forgetfulness, confusion, and muscle cramps. More severe symptoms include seizures, obtundation, coma, and respiratory arrest.

Patients with severe symptoms warrant emergent management. Rapidly increasing the serum sodium is paramount to prevent permanent neurologic damage. The goal should be to increase the serum sodium by 2 to 4 mEq/L until symptoms abate as rapidly as possible.[29] This can be achieved by infusion of 100 mL boluses (or 5 mL/kg in children) of 3% HTS up to a maximum of 3 boluses.[30] If HTS is not readily available, 8.4% sodium bicarbonate ampules from the code cart can be used as an acceptable alternative. Ampules of 50 mL of 8.4% sodium bicarbonate contain almost 6% sodium and is effectively equal to 100 mL of HTS. If the serum sodium increases adequately and symptoms persist, consider another cause other than hyponatremia.

Experts have long debated the appropriate correction rate for mildly symptomatic hyponatremia. From multiple studies, it has been well established that correction of the sodium level should not occur quicker than 10 to 12 mEq/L per 24 hours, with some conservative experts stating the correction should not occur faster than 6 mEq/L per 24 hours to prevent complications.[31,32] Both United States and European guidelines now recommend a correction limit of 10 mmol/L per day regardless of chronicity and advise frequent monitoring response to treatment.[31,33] Where they diverge is in their recommendations for managing overcorrection. Although both recommend using vasopressin or free water to address overcorrection, they differ on when one should intervene. Per the United States guidelines, if the limit is reached in a patient whose baseline sodium was greater than 120 mmol/L, then correction is likely unnecessary. If the baseline was less than 120 mmol/L, then the clinician ought to relower the sodium. The European guidelines suggest relowering if the limit of correction has been surpassed, regardless of initial baseline concentration.[31]

Both guidelines recommend the use 3% HTS for the treatment of symptomatic hyponatremia. The United States guidelines still holds slightly differing intervention recommendations in terms of acute versus chronic hyponatremia; however, they recommend for mild symptoms 3% HTS as a continuous infusion at 0.5 to 2 mL/kg per hour.[31] Comparatively, the European guidelines recommend 3% HTS bolus 150 mL over 20 minutes once.[31] There is a recent study that compares bolus therapy and continuous infusion, and it found that both were equally effective but that the bolus therapy had a lower incidence of overcorrection requiring relowering therapy.[34]

Except in hypovolemic hyponatremia, both the US and European guidelines recommend fluid restriction as first-line therapy for management of chronic euvolemic and hypervolemic hyponatremia. Vaptans, which block vasopressin type 2 receptors to cause free water loss or aquaresis, is suggested as second-line therapy for SIADH (or euvolemic hyponatremia) and hypervolemic hyponatremia in the United States.[33] However, vaptans may cause liver toxicity as well as overcorrection.[31,35,36] For this reason, the European guidelines do not recommend it for moderate hyponatremia and recommend against it in severe cases and in hypervolemic hyponatremia. Instead, the European second-line for SIADH includes loop diuretics. Nevertheless,

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in most patients, correction of the underlying medical conditions will usually correct the hyponatremia.[31]

In chronic hypovolemic hyponatremia, volume expansion is the mainstay of treatment. Fluid resuscitation must be initiated and any underlying problem causing the volume depletion must be corrected. As the intravascular volume is restored, ADH secretion drops, renal function improves, and excess free water can be excreted.[31] Most commonly, the initial fluid used is 0.9% normal saline. Guidelines suggest the clinician may use isotonic saline alone (United States) or also balanced crystalloid (European).[31,33]

Disposition depends on the patient's symptoms. If the patient is severely symptomatic, they warrant ICU admission. Mild symptoms with moderate to severely decreased serum sodium levels warrant admission to the floor depending on the hospital protocols for serum sodium levels. Asymptomatic patients or patients with mildly decreased sodium levels can often be discharged with close follow-up.

Complications

Cerebral edema

As stated previously, patients with severe hyponatremia can develop cerebral edema because of water moving from the extracellular to the intracellular compartment because of a difference in effective osmolality between brain and plasma. This usually occurs when hyponatremia develops rapidly, and the brain has had too little time to adapt to its hypotonic environment.[31] Patients particularly at risk for this include postmenopausal women, women on thiazide diuretics, children, psychiatric patients with polydipsia, and hypoxemic patients.[32] Once treatment of hyponatremia has commenced, it is important to watch electrolyte levels closely, checking every 2 hours if needed to help guide medical therapy. All these measures are to prevent the dreaded complication known as osmotic demyelination syndrome.

Osmotic demyelination syndrome

ODS is the iatrogenic irreversible clinical syndrome of neurologic symptoms that occurs after too rapid a correction of serum sodium.[19] This severe complication is caused by exceeding the safe limits of serum sodium correction, which as stated previously, is 10 to 12 mEq/L in a 24-hour period.[19] This syndrome is postulated to be caused by rapid swelling of the brain and nervous system tissue parenchyma during fluid shifts because the tissue cannot adapt quickly enough to the changing osmolality. Symptoms of osmotic demyelination syndrome include fluctuating levels of consciousness or confusion, behavioral changes, dysarthria, and dysphagia, with the worst symptoms being seizures, "locked-in syndrome" (devastating permanent condition where all the voluntary muscles are paralyzed aside from the eyes), and death. This condition can take anywhere from 2 to 6 days to develop and, thus, will often not be clinically apparent while the patient is still in the emergency department. Therefore, it is incumbent on the emergency clinician to be very meticulous when it comes to the treatment of hyponatremia to prevent rapid overcorrection to prevent this condition.

HYPERNATREMIA

Defined as a serum sodium of greater than 145 mmol/L, hypernatremia is less common than hyponatremia, and it is most commonly due to excess water loss. However, it can also be caused by the intake of salt without water or the administration of HTS solutions. The vast majority is diagnosed in hospitalized patients but, according to one population-based study, 0.2% of patients with hypernatremia were diagnosed in the emergency department.[37] The clinical manifestations of acute hypernatremia are

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caused by an increase in serum sodium and osmolality causing water movement out of brain cells and into the extracellular space subsequently causing brain cells to shrink. Similar to hyponatremia, hypernatremia is associated with a higher mortality, especially with other comorbid conditions.[38]

Presentation

The symptoms of acute hypernatremia are most attributable to the rapidity of change of the serum sodium levels, not necessarily the overall sodium excess (or free water deficit). The cause of hypernatremia is usually evident on clinical presentation and, as stated previously, it most often occurs when water loss outpaces replacement.[3] Alternatively, acute hypernatremia can be induced by large amounts of salt ingestion or it can be iatrogenic in the case of administration of hypertonic sodium containing solutions. The most common initial symptoms include thirst, dry mucous membranes, and decreased urine output. Again, the central nervous system is the most sensitive to changes in serum osmolality and is usually the most affected. Therefore, as the condition progresses, more pronounced neurologic signs and symptoms may emerge, including, restlessness, irritability, confusion, lethargy, muscle twitching, and seizures. Severe hypernatremia can lead to neurologic complications such as altered mental status, hallucinations, coma, and even death, if left untreated. The shrinking of brain cells can cause intracerebral hemorrhage because of tearing of cerebral blood vessels. The severe manifestations of hypernatremia usually are not present until the serum sodium increases more than 158 mEq/L and values greater than 180 mEq/L are associated with a very-high mortality rate.[18]

Evaluation and Diagnosis

As with hyponatremia, the first step when evaluating a patient with hypernatremia is to determine the cause and then the volume status of the patient. See **Table 2** for common causes of hypernatremia. Hypovolemic hypernatremia is usually caused by decreased water intake in patients with limited access to free water; however, it can also be due to the inability to detect or respond to the sensation of thirst. Primary adipsia or hypodipsia is caused by damage to the hypothalamic osmoreceptors. Patients with hyperglycemic hyperosmolar syndrome are clinically dehydrated and have a relative hypernatremia due to the hyperglycemia-induced eunatremia on laboratory work. Correction of sodium based on the glucose levels will often reveal the true sodium level. Hypervolemic hypernatremia is usually iatrogenic in nature from the administration of hypertonic solutions or from the improper preparation of solutions for enteral or parenteral feeding or peritoneal dialysis. Euvolemic hypernatremia most often develops in diabetes insipidus (DI) in which the body does not respond to ADH either centrally or peripherally. Therefore, the patient continues to make maximally dilute urine with low urine osmolality. Causes

Table 2
Common causes of hypernatremia

Hypervolemic hypernatremia	Euvolemic hypernatremia	Hypovolemic hypernatremia
Hypertonic solution administration	Diabetes insipidus	Dehydration due to decreased water intake
		Prolonged hyperglycemia
		Hyperglycemic hyperosmolar syndrome
		Osmotic diarrhea
		Hyperaldosteronism

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of DI are divided into 2 categories: central and nephrogenic. Central DI, also known as arginine vasopressin deficiency, can be caused by trauma, pituitary surgery, and malignancies but is most commonly idiopathic. Arginine vasopressin deficiency is characterized by a decrease in the release of ADH resulting in a variable degree of polyuria. About 30% to 50% of cases are idiopathic and are associated with the destruction of the hormone secreting cells in the hypothalamic nuclei.[39] Nephrogenic DI, also known as arginine vasopressin resistance, is most commonly caused by renal disease, medications, and genetic disorders.

Management

The approach to the treatment of hypernatremia is relatively straightforward given that the range of causes is less than that for hyponatremia. The most important factors to consider are whether the increase was acute or chronic, and the severity of symptoms.

Acute hypernatremia is defined as a serum sodium level of greater than 145 mEq/L, which develops during a period of less than 48 hours. If it is known that the hypernatremia developed during a period of greater than 48 hours, or if the duration is unknown, then it is considered chronic.[40]

Unstable patients should have their hypernatremia corrected during a minimum of 48 hours to decrease the likelihood of seizures, permanent brain damage, or even death, which can occur in the setting of rapid correction with hypotonic fluids. The serum sodium should not decrease by more than 8 to 15 mEq/L in a given 8-hour period.[41] The use of isotonic fluids (0.9% normal saline or lactated ringers) to restore plasma volume is optimal because it is still likely hypotonic compared with the patients' serum and is less likely to result in too rapid of overcorrection. However, the serum sodium must be closely monitored to prevent overcorrection, leading to additional neurologic problems.[6] If the correction does occur too rapidly, or if the patient begins to show signs of cerebral edema or hyponatremia, the fluid replacement should immediately stop and the patient should be treated for hyponatremia, with fluid restriction.

Stable patients will have less symptoms and a lower associated mortality because it relates to their sodium levels. In these chronic cases, the brain goes into self-preservation mode and produces idiogenic osmoles that allows relative maintenance of brain cell hydration. In an asymptomatic patient, correction of the serum sodium level should occur slowly, at a rate of no greater than 0.5 mEq/L/h and no more than 8 to 15 mEq/L per day.[40] The specific treatment of hypernatremia is tailored to the nature of the cause. If the patient is asymptomatic and has normal renal function, then the clinician can wait for natural excretion. If the patient's renal function is impaired, then excess sodium may need to be removed by dialysis. In patients with adipsia or hypodipsia, patient, caregiver, and family education is of paramount importance.

Complications

Cerebral edema

Rapid correction of hypernatremia can lead to a shift of water into brain cells, causing cerebral edema. This can result in neurologic symptoms such as seizures, altered mental status, focal neurologic deficits, and even coma. To prevent this, a gradual and controlled correction of sodium levels is important.

Osmotic demyelination syndrome

Overly rapid correction of sodium levels, particularly in cases of chronic hypernatremia, can cause the destruction of myelin in the brainstem, leading to a condition called central pontine myelinolysis or osmotic demyelination syndrome. This condition can

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result in a range of neurologic symptoms, including paralysis, dysarthria, dysphagia, and other motor deficits.

Fluid overload

Inappropriate administration of fluids or excessive water replacement can lead to fluid overload, especially in patients with compromised cardiac or renal function. This can result in pulmonary edema, congestive heart failure, or exacerbation of existing edematous conditions.

Electrolyte imbalances

Correcting hypernatremia can affect the balance of other electrolytes, such as potassium and magnesium. Rapid shifts in electrolyte levels can lead to cardiac arrhythmias, neuromuscular abnormalities, and other complications.

To minimize the risk of these complications, it is crucial to closely monitor the patient's serum sodium levels, fluid balance, and clinical status during the treatment of hypernatremia. The correction of sodium levels should be gradual and tailored to the individual patient's needs.

SUMMARY

Sodium imbalances are a common occurrence in the emergency department. Although recognition and diagnosis are relatively straightforward, discovering the cause and management should be approached systematically. The most important history items to ascertain is if the patient has symptoms and how long this imbalance has taken to develop. Treatment rapidity depends on severity of symptoms with the most rapid treatment occurring in only the severely symptomatic. Overcorrection has dire consequences and must be approached in a careful and systematic fashion in order to prevent these devastating consequences.

CLINICS CARE POINTS

- Evaluating the patient's volume status is critical to helping ascertain the possible cause for the sodium imbalance.
- Rapidity of the development of the patient's sodium imbalance and if the patient has symptoms will often drive the management of the patient.
- Patients with severe symptoms require rapid treatment to prevent permanent neurologic sequelae.
- If HTS therapy is not readily available, you can use 8.4% sodium bicarbonate as an acceptable alternative.
- Preventing overcorrection should be foremost on the emergency clinician/s mind when managing sodium imbalance. Have a low threshold to check sodium levels more frequently while correcting.

DISCLOSURE

The authors have no financial disclosures.

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